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REVIEW ARTICLE

Periodontal vaccine: A therapeutic modality on the horizon?

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Abstract Periodontal diseases with their poly microbial etiology, are a major cause of tooth mortality in the adult population. Current treatment modalities have resulted only in arresting the disease progression but have not cured the disease completely, nor do they prevent the recurrence. Hence there is a need for more sophisticated therapeutic modalities which may include vaccines targeting putative periodontal pathogens. No periodontal vaccine trials have been successful in satisfying all requirements of an ideal periodontal vaccine. Periodontal vaccines could emerge as an adjunct to mechanical therapy in future.
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1. Introduction

In the current paradigm of periodontal disease, the periodontal pathogens are essential for disease initiation, however, the extent and severity of tissue destruction largely depend on the nature of the host microbial interactions. Periodontal diseases are immune inflammatory responses induced by microorganisms in dental plaque which, harbored within a susceptible periodontium contributes to tissue destruction, bone loss and eventually tooth loss.

Specific molecules have been recognized that signal periodontal tissue destruction as the inflammatory response develops. They can be broadly divided into (a) microbial virulence factors (b) those derived from host immune inflammatory response. Bacteria are important because they drive and perpetuate the inflammation but the great majority of tissue breakdown results from host inflammatory processes. Microbial virulence factors include (a) lipopolysaccharides (b) bacterial enzymes and noxious products (c) microbial invasion strategies (d) fimbriae (e) bacterial deoxyribonucleic acid and extracellular deoxyribonucleic acid. The host derived inflammatory mediators can be divided as (a) cytokines (b) prostaglandins and (c) matrix metalloproteinases.

Till date, no preventive modality exists for periodontal disease and treatment rendered is palliative. The availability of periodontal vaccine would not only prevent or modulate the course of periodontal diseases, but also enhance the quality of life of people for whom periodontal treatment cannot be obtained easily.

2. Bacterial etiology

Acceptance of the specific plaque hypothesis was spurred by the recognition of Actinobacillus actinomycetemcomitans as a pathogen in localized aggressive periodontitis[1].

Porphyromonas gingivalis, *A. actinomycetemcomitans*, Treponema denticola, *Tannerella forsythia* were implicated as the key pathogens in the etiology of periodontal disease. According to Loesche WJ in 1976, the specific plaque hypothesis states that only certain plaque is pathogenic, and its pathogenicity depends on the presence or increase in the specific microorganisms[2].

With the rapid growth of microbial genome sequencing and bioinformatics analysis tools, we have the potential to examine all the genes and proteins from any human pathogen[3]. This technique has the capability to provide us with new targets for anti-microbial drugs and vaccines. However, to realize this, potential new bioinformatics and experimental approaches for the selection of these targets from the myriad of available candidates are required.

3. Host response in periodontal disease

The host defense against periodontopathogenic bacteria comprises innate and acquired immunity. Saliva, GCF and the keratinocytes are some of the key agents that play a key role in innate immune response[4,5]. Neutrophils are the primary leukocytes to act in innate immune response. Macrophages and dendritic cells are also important innate immune cells which express pattern recognition receptors (PRRs) that interact with the specific molecular structures on microorganisms called microbe associated molecular patterns (MAMPs) to signal immune responses[6,7]. However innate immune response is nonspecific hence results in excessive host tissue damage without effective antigenic clearance[8,9]. Adaptive immunity has evolved to provide a focused and intense defense against infections that overwhelm innate immune responses in the tissues[10–12]. Adaptive immunity is slower and reliant on complex interactions between antigen-presenting cells and T and B lymphocytes, cytotoxic T cells and antibodies[13–15]. The increase in antibody titer or antigen specific T–cells resulting from an exposure of a host to an antigen for the first time is referred to as the primary response. The secondary response develops after a subsequent exposure to that same antigen. Because of the generation of memory, the secondary response

- Is more rapid in onset
- Is longer in duration
- Is greater in strength due to higher titers
- For B cells may have greater specificity, against the antigen compared with the primary response.

The expanded pool of memory cells provides a reservoir of cells that is sustained for years by constant stimulation of antigen maintained by follicular DCs (Dentritic Cells). The primary response takes slightly more than one week to become measurable and biologically or clinically useful. Secondary responses are clinically measurable within 1 to 3 days and are so effective that an individual may not be aware of the infection. The immune response to pathogenic microorganisms involves the integration at the molecular, cellular, and organ level of elements often categorized as being part of the innate immune system or the adaptive immune system. Periodontopathogens have however developed mechanisms to inhibit and evade cell-mediated and humoral immune responses[16–18].

4. Vaccine

Vaccine is the name applied generally to a dead or attenuated living infectious material introduced into the body with the object of increasing resistance or eliminating the disease. Vaccination is the development of immunity, or resistance to

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